Sleep Medicine 15 (2014) 203–208

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Sleep and risk for high blood pressure and hypertension in midlife women: the SWAN (Study of Women's Health Across the Nation) Sleep Study

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ARTICLE INFO

Article history:
Received 28 March 2013
Received in revised form 1 November 2013
Accepted 5 November 2013
Available online 20 November 2013

Keywords:
Hypertension
Sleep duration
Sleep stages
Polysomnography
Women
Epidemiology

ABSTRACT

Objective: Inadequate self-reported sleep is related to high blood pressure (BP). Our study investigated cross-sectional and longitudinal relationships between poor sleep measured by in-home polysomnography (PSG) and BP.
Methods: Midlife participants (132 black, 164 white, and 59 Chinese) were from the SWAN (Study of Women's Health Across the Nation) ancillary sleep study. In-home PSG measured sleep apnea, duration, efficiency, and electroencephalogram (EEG) total delta and beta power during nonrapid eye movement (NREM) sleep. Women subsequently were followed annually for 4.5 (1–7) years for BP and hypertensive status (>140/90 mmHg or use of antihypertensive medication). Covariates were age, race, site, and educational attainment, with time-covariates of BP medications, body mass index, diabetes mellitus (DM), cigarette smoking, and menopausal status.
Results: Sleep duration and efficiency were unrelated to BP cross-sectionally or longitudinally in multivariate models. Women with higher total beta power were more likely to be hypertensive at the time of the sleep study; women with lower total delta power were more likely to show increases in diastolic BP (DBP) and to be at risk for incident hypertension across follow-up.
Conclusions: Low NREM delta power may be a risk factor for future hypertension. Quantitative EEG measures are worthy of future investigations of hypertension risk.

1. Introduction

Poor sleep may contribute to the development of high blood pressure (BP) [1]. Self-reported sleep duration is the most frequently investigated dimension in epidemiologic studies. In the First National Health and Nutrition Examination Survey, men and women who reported typical sleep durations of ⩾5 h per night were at increased risk for incident hypertension over the 8- to 10-year follow-up period, after adjusting for sociodemographic factors, health behaviors, obesity, and diabetes mellitus (DM) [2]. Among British civil servants, women reporting ⩽5 h of sleep were at increased risk for incident hypertension over 5 years [3]. The association did not remain significant in multivariate analyses and was not present in men. In the Sleep Heart Health Study, men and women who reported typical sleep durations of <6 h were at elevated risk for prevalent hypertension [4]. However, in both the Western New York Healthy Study and the Heinz Nixdorf Recall Study women reporting <6 h of sleep were at elevated risk for prevalent hypertension, but no associations were observed in men [5,6].

Studies using methods other than self-report have suggested a more complex picture. In the Rotterdam Study, polysomnography (PSG)-measured sleep duration was unrelated to incident hypertension in elderly men [7]. In contrast, shorter actigraphy-measured sleep duration was associated with higher levels of systolic BP (SBP) and diastolic BP (DBP) and increased odds of hypertension in the Coronary Artery Risk Development in Young Adults (CARDIA) cohort of middle-aged adults; these associations were partially attenuated after adjustment for health and additional sleep factors [8]. In addition, lower sleep maintenance (i.e., proportion

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http://dx.doi.org/10.1016/j.sleep.2013.11.002

of time asleep between initial sleep onset and final awakening) also was associated with increases in DBP over time. Two Swedish studies found that those with hypertension had shorter and lower efficient sleep compared to normotensive patients measured by ambulatory PSG [9,10]. Beyond sleep duration and continuity, sleep can be characterized by other dimensions, including sleep stages and quantitative electroencephalogram (EEG) characteristics such as power in different frequency bands. Beta power during nonrapid eye movement (NREM) sleep is an index of hyperarousal, which is associated with insomnia symptoms [11,12]. Deep NREM sleep stage is characterized by high amounts of delta EEG power. It is associated with better performance and learning [13,14]. It also is associated with metabolic and hormonal variation and decreased sympathetic nervous system activity and increased parasympathetic activity during the night, which are associated with lower BP [15]. The only epidemiologic study regarding slow-wave sleep (SWS) in relation to incident hypertension yielded intriguing results. Among elderly men who had in-home unattended PSG, lower percent of SWS predicted incident hypertension across 3.4 years [16]. This relationship was not confounded by sleep-disordered breathing (SDB). The objective of our study was to examine the association between risk for high BP and PSG-derived sleep characteristics: sleep duration, efficiency, and total delta and beta power during NREM sleep. In the Study of Women's Health Across the Nation (SWAN), an ancillary study measured sleep by in-home PSG for three nights in midlife women; BP was measured annually. We tested the hypotheses that women who had lower total delta and higher total beta power, shorter sleep duration, and less efficient sleep would have higher BP and be at risk for hypertension concurrently and longitudinally across 1–7 years.

2. Methods

2.1. Participants

Participants were black ($n = 139$), white ($n = 172$), and Chinese ($n = 59$) women enrolled in the SWAN Sleep Study [17]. SWAN is a community-based 7-site study of aging in midlife women [18]. Eligibility criteria for the longitudinal cohort were (1) those ages 42–52 years, (2) those with an intact uterus, (3) those with at least one menstrual period not using exogenous hormones (e.g., birth control, hormone therapy) in the 3 months prior to the baseline interview, and (4) those who self-identified with the designated race/ethnic groups of the site. The institutional review boards at all seven participating sites approved the study protocol.

Starting in year four of the core SWAN study at four sites (Chicago, IL; Detroit area, MI; Oakland, CA; and Pittsburgh, PA), potential eligibility for the SWAN Sleep Study was determined during the participants' annual SWAN visit. Efforts were focused on recruiting women who were pre- and perimenopausal and excluded women who had hysterectomy (<1%) or who reported using hormone therapy (approximately 23% of the cohort). During the final year of recruitment (year seven of SWAN), eligibility criteria were relaxed to allow natural postmenopausal women into the protocol. Additional exclusions for the sleep study were current chemotherapy or radiation, current oral corticosteroid use, regular night-shift work, regular consumption of >4 alcoholic drinks per day, and noncompliance with core SWAN procedures (i.e., missed >50% of annual visits, refused annual visit blood draw). All potentially eligible women were approached regarding participation at the four sites. Of those who were approached, 30% declined to participate primarily due to protocol burden. The SWAN Sleep Study enrolled 370 white, black, and Chinese participants. These participants did not differ from core SWAN participants at these sites at core visit year five regarding self-reported sleep quality, race/ethnicity, self-reported health status, depressive symptoms, or physician-diagnosed hypertension or DM. Participants did have a slightly higher body mass index (BMI) than nonparticipants. Of the 370 participants, 355 had both PSG data and BP or hypertension data, with 330 having delta and beta power data. Informed consent was obtained in accordance with approved protocols and guidelines of the institutional review board at each participating institution. Participants received monetary compensation for their participation.

2.2. Protocol and assessments

2.2.1. Blood pressure

At each visit throughout SWAN, SBP and DBP were manually measured twice by trained and centrally certified technicians. Readings were taken on the right arm with a minimum of 2-min rest periods between readings. The participant was seated with feet flat on the floor for at least 5 min prior to the measurement. She had not smoked or consumed any caffeinated beverage within 30 min of BP measurement. Appropriate cuff size was determined based on arm circumference. The two sequential BP values were averaged. At each visit, women were queried about current medication use including hypertension medications. In our analysis, hypertension was defined as SBP of $\geqslant 140$ mmHg or DBP of $\geqslant 90$ mmHg or any BP medication use. Baseline BP for our report was considered to be BP at the SWAN visit closest in time to the sleep studies.

2.2.2. Sleep

Unattended PSG sleep studies were conducted in participants' homes for three consecutive nights; occasionally one of the nights was repeated due to equipment problems. Staff visited participants in their homes on each night of PSG studies to apply electrodes and calibrate monitors. Participants slept in their own beds under their usual circumstances at their habitual sleep and wake times, as determined by self-report. On rising in the morning, participants removed study equipment and turned off the recorder.

PSG sleep data were collected with Vitaport-3 (TEMEC VP3™) ambulatory monitors and software. Signals collected on each study night included bilateral central referential EEG channels (C_3 and C_4, referenced to A_1–A_2), electrooculogram, submentalis electromyogram, and electrocardiogram. Additional signals were collected on the first night of sleep studies for the assessment of SDB (nasal pressure and oral-nasal thermistors, pulse oximetry, and abdominal and thoracic excursion as measured by inductance plethysmography to reflect ventilatory effort). Sleep stage scoring was conducted using Harmonie software. Signals were filtered during acquisition (0.3-Hz high-pass filters and 100-Hz low-pass filters, with 60-Hz notch). Modified periodograms were computed using the fast Fourier transform (FFT) of nonoverlapping 4-s epochs of the sleep EEG aligned with the 20-s visually scored sleep data with locally developed software, which included a validated automated artifact rejection routine [17]. Although FFT assumes a stationary signal, which is never completely true in the EEG, the large number of 4-s epochs per participant during NREM sleep produces stable estimates of power. FFT also can lead to inaccurate assessments of power due to frequency leakage. Hamming window (v^2/Hz and zero overlap) on nonoverlapping 4-s epochs was used to reduce spectral leakage. Quality assurance assessments, scoring, and processing of all sleep study records were performed at the University of Pittsburgh Neuroscience, Clinical and Translational Research Center. The coders were trained PSG technologists with established reliability (intraclass correlation coefficients for wake, rapid eye movement [REM], and NREM sleep periods were each above .90), who were blind to participant characteristics. Sleep was scored in 20-s epochs using Rechtschaffen and Kales criteria

[19,20], as data were collected prior to the updated American Academy of Sleep Medicine *Manual of Scoring Sleep and Associated Events*.

Measures of sleep duration and efficiency were based on averages of sleep study nights two and three, whereas SDB was assessed on night one only. Total sleep duration was calculated as total minutes of all stages of sleep from sleep onset to morning awakening converted into hours and averaged across nights. Sleep efficiency (total sleep time/time spent in bed $\times$ 100) was used to quantify sleep continuity. Power in the delta (0.05–4.01 Hz) and beta (16.0–32.0 Hz) was calculated as total power within each band. For the majority of participants (69%), power was averaged across both study nights; we previously reported that one night is sufficient to derive reliable estimates of delta and beta power [21]. Few EEG epochs were discarded due to artifact rejection routines (mean [standard deviation {SD}], 4.6 [3.3] min NREM sleep). To normalize their distributions, NREM total delta and total beta power were log transformed and sleep efficiency was inverse-log transformed (i.e., log [100 – sleep efficiency + 1]). In our sample, NREM delta and beta power were associated with percent SWS (r = 0.71 and 0.18; P < .001, respectively) and were associated with one another (r = 0.26; P < .001).

The Pittsburgh Sleep Quality Index (PSQI) was administered on day four of the protocol. The following question was embedded in the questionnaire: during the last month, how many hours of actual sleep did you get at night? [22]. This question is comparable to questions about self-reported sleep in a number of epidemiologic studies.

2.2.3. Potential covariates

SDB was quantified by the apnea–hypopnea index (AHI) (number of apneas + number of hypopneas/total sleep time [19]) and was log transformed for analyses. BMI was measured in clinic as part of the annual SWAN core examination. Race was established at the first visit by self-identification (non-Hispanic white, Chinese, or black). Based on self-reported menstrual bleeding patterns, participants were categorized as premenopausal, early perimenopausal (menses in last 3 months but irregular), late perimenopausal (no menses for 3–11 months), and postmenopausal (no menses for at least 12 months or hysterectomy or bilateral oophorectomy, of which 16 participants began hormone use after becoming postmenopausal.) Educational status was classified into two groups, those with a 4-year college degree or more and those with less than a college degree. Cigarette smoking was assessed by daily diary reports of smoking during the study (any nicotine use was coded as yes). DM was defined as self-reported DM in combination with self-reported oral or injected medication for DM. Medication use was recorded at Sleep Study protocol inception and was coded according to the World Health Organization Anatomic, Therapeutic, and Chemical Classification System (http://www.whocc.no/atcddd). For our report, medications were based on diary records at the time of the sleep studies. Medications that affect sleep, though not necessarily prescribed for sleep problems, and antihypertensive medications were covariates. Center for Epidemiologic Studies-Depression scale (CES-D) was completed annually. This 20-item scale is widely used in epidemiologic studies and has been used in diverse ethnic groups [23,24]; it was coded as $\geqslant$16 yes or no format. Age and site also served as covariates. In preliminary multivariate models, CES-D, logged AHI scores, and medications that could affect sleep were nonsignificant in all models. Two women did not have CES-D scores, 18 did not have AHI scores, and six did not have data regarding medications that may affect sleep. Thus we decided not to use these variables as covariates, as we did not want to substantially reduce the sample size. Correlation between BMI and logged AHI scores was r = .23. Thus the covariates used in our analyses were age, site, race/ethnicity, educational attainment, menopausal status, BMI (continuous), DM, and smoking status; baseline BP and antihypertensive medications were used as covariates noted below.

2.3. Data analyses

Cross-sectional associations between sleep measures and BP were assessed by linear regression analyses. Models were first adjusted for age only and then for the full set of covariates (site, race/ethnicity, BP medication use, education, BMI, smoking, menopausal status, and DM [yes/no]). The associations between hypertension and sleep measures were estimated by logistic regression analyses with the same covariate adjustment excluding BP medication use, which was part of the definition of hypertension.

Longitudinal associations between sleep measures with sleep study data collection beginning during SWAN visit four and change in BP from the sleep study to each follow-up visit through SWAN visit 12 were estimated by mixed models with random intercept. The same covariate adjustments as with the linear regression models were made (i.e., race/ethnicity, site, education, plus BP at time of sleep study, and with age, BP medication, menopausal status, BMI, smoking, and DM as time-varying covariates in the mixed models). An interval-censored survival analysis was applied to examine the relation between incident hypertension and sleep measures in the 256 women who were not classified as hypertensive at the time of the initial sleep study. The models were first adjusted for age only and then site, race/ethnicity, education, BMI, smoking, menopausal status, and DM at the SWAN study visit that was closest to the sleep study. In supplementary multivariate analyses, we tested for the interactions between sleep variables and BMI and race/ethnicity; none of the variables were significant. Analyses were performed with SAS version 9.1.3 (SAS Institute, Cary, NC). All models were two sided (α, .05).

3. Results

A total of 355 (black [n = 132], white [n = 164], and Chinese [n = 59]) women were included in our analyses, with a baseline mean age of 51.5 years (SD, 2.2). Approximately half of the individuals were white (46%) and college educated (52%). At the time of sleep study, the majority of women were in the menopausal transition, free of hypertension, and were not using any BP medication or medication that may affect sleep (Table 1). Approximately 10% were current smokers. On average the women slept 6.4 h a night and had sleep efficiency of 84.3% based on PSG. Women were followed for an average of 4.5 years (SD, 1.4; range, 1–7) after the sleep study. Participants' BP on average did not change over the 7-year follow-up period and BP changes did not vary by ethnic group (P > .3). At the time of the sleep study, the 99 women classified as hypertensive were more likely to be older, less educated, black, smokers, and have DM; they also had elevated AHI and BMI scores compared to normotensive participants (Table 1). Of the 256 women free of hypertension at the sleep study, 247 had follow-up BP or antihypertensive medication data, with 64 (black [n = 29], white [n = 29], Chinese American [n = 6]) becoming hypertensive during the follow-up period.

3.1. Sleep duration and efficiency

In age-adjusted cross-sectional models, less efficient sleep was related to higher SBP (P < .0001) and less efficient sleep (odds ratio [OR], = 2.58 [1,47.4.50]) and shorter sleep duration (OR, = 0.68 [0.53,0.87]) were related to increased risk for hypertension prevalence (see Table 1 for unadjusted means of sleep variables by hypertensive status). Further analysis showed that the addition

Table 1
Sample description by hypertensive status at the time of the sleep study.

Mean (SD) or *n* (%)	Full sample 355	Normotensive 256	Hypertensive 99	*P* value
Mean (SD) age, y	51.5 ± 2.2	51.4 (2.2)	51.9 (2.2)	.03
Mean (SD) BMI kg/m^2	29.9 ± 7.5	28.3 (6.7)	34.3 (7.7)	<.0001
n (%) BMI				
>30 kg/m^2	148 (41.7)	85 (33.2)	63 (63.6)	<.0001
⩽30 kg/m^2	207 (58.3)	171 (66.8)	36 (36.4)	
n (%) Race/ethnicity				
Black	132 (37.2)	71 (27.7)	61 (61.6)	<.0001
Chinese	59 (16.6)	50 (19.5)	9 (9.1)	
White	164 (46.2)	135 (52.7)	29 (29.3)	
n (%) Current smoker	36 (10.1)	16 (6.3)	20 (20.2)	<.0001
n (%) Menopausal status				
Postmenopause	76 (21.4)	47 (18.4)	29 (29.3)	.03
Late perimenopause	78 (22.0)	52 (20.3)	26 (26.3)	
Early perimenopause	182 (51.3)	142 (55.5)	40 (40.0)	
Premenopause	19 (5.4)	15 (5.9)	4 (4.0)	
Education				
<4-year college degree	169 (48.3)	110 (43.3)	59 (61.5)	.002
⩾4-year college degree	181 (51.7)	144 (56.7)	37 (38.5)	
n (%) AHI				
<5	163 (47.9)	128 (51.6)	35 (38.0)	.02
5– < 15	129 (37.9)	92 (37.1)	37 (40.2)	
⩾15	48 (14.1)	28 (11.3)	20 (21.7)	
n (%) DM	21 (5.9)	6 (2.3)	15 (15.2)	<.0001
n (%) CES-Depression				
<16	28 (84.2)	212 (86.5)	71 (78.0)	
⩾16	53 (15.8)	33 (13.5)	20 (22.0)	.06
Mean (SD) SBP mmHg	116.3 ± 16.9	113.0 (12.3)	134.0 (18.3)	<.0001
Mean (SD) DBP mmHg	73.5 ± 9.8	71.6 (8.2)	78.7 (11.9)	<.0001
n (%) Blood pressure medication use	80 (22.9)	N/A	80 (85.1)	N/A
n (%) Medication use affecting sleep	95 (27.2)	68 (27.1)	27 (26.6)	.93
Mean (SD) PSG sleep efficiency	84.4 (8.2)	85.3 (7.1)	82.0 (10.3)	.001
Mean (SD) PSG sleep duration (h)	6.4 (1.0)	6.5 (0.9)	6.1 (1.1)	.002
Mean logged (SD) NREM sleep total delta power (μv^2/Hz)	5.04 (0.45)	5.05 (0.42)	5.02 (0.51)	.55
Mean logged (SD) NREM total beta power (μv^2/Hz)	1.17(0.41)	1.13 (0.39)	1.29 (0.43)	.001

Abbreviations: SD, standard deviation; *n*, number of patients; y, years; BMI, body mass index; AHI, apnea–hypopnea index; DM, diabetes mellitus; CES, Center for Epidemiologic Studies; SBP, systolic blood pressure; DBP, diastolic blood pressure; PSG, polysomnography; NREM, nonrapid eye movement.
P value from *t* tests or χ^2 testing differences between normotensive and hypertensive women with no covariates.

of race/ethnicity to the age-adjusted models resulted in nonsignificant effects for duration and efficiency.

In the longitudinal analyses, adjusting for age and baseline BP, less efficient sleep was related to increases in SBP and DBP ($P \leqslant .01$) and increased risk for incident hypertension across the follow-up period (OR, 2.26 [95% confidence interval {CI}, 1.19–5.86]), whereas shorter sleep duration was related to a larger increase in DBP ($P = .03$). None of the multivariate models, including either cross-sectional or longitudinal models, approached statistical significance for either sleep efficiency or duration. Secondary analyses for sleep duration examined if women with less than 6 h were at increased risk for incident hypertension or BP change; none of the multivariate analyses were significant. Answers to the self-reported sleep question from the Pittsburgh Sleep Quality Index also were unrelated to BP or hypertensive status in either categorical or continuous analyses ($P > .15$).

3.2. NREM total beta and delta power

In the cross-sectional age-adjusted analyses, higher beta power was associated with higher SBP ($P < .01$) and an increased risk for prevalent hypertension (OR, 2.70 [95% CI, 1.47–4.96]). In the multivariate models, the association of higher beta power and prevalent hypertension remained significant (Table 2). Beta power was unrelated to BP change or hypertensive status in the longitudinal analyses, regardless if it was age adjusted only or fully adjusted.

Table 2
Multivariate associations between logged total delta and total beta power during nonrapid eye movement sleep and blood pressure and hypertension.

Cross-sectional†	SBP Estimate (SE)	DBP Estimate (SE)	Hypertension Hazards ratio (95% CI)
Delta	−2.49 (1.93)	−1.61 (1.25)	0.94 (0.46, 1.93)
Beta	1.85 (2.03)	0.03 (1.31)	2.34 (1.07, 5.10)*
Longitudinal††	**Change SBP Estimate (SE)**	**Change DBP Estimate (SE)**	**Incident hypertension Hazards ratio (95% CI)**
Delta	−1.94 (1.20)	−1.86 (0.67)**	0.49 (0.32, 1.01)*
Beta	−1.26 (1.25)	−0.48 (0.69)	1.09 (0.61, 2.76)

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; SE, standard error; CI, confidence interval.
Note: $n = 325$ for hypertension prevalence and $n = 318$ for BP in cross-sectional analyses; $n = 299$ for hypertension incidence and $n = 309$ for BP change in longitudinal analyses.
* $P \leqslant .052$.
** $P < .01$.
† Adjusted for age, site, race/ethnicity, blood pressure (BP) medication, body mass index (BMI), education, diabetes mellitus (DM), and smoking status.
†† Adjusted for age, site, race/ethnicity, BMI, DM, smoking status, education, and baseline BP in change analyses; without baseline BP in incident hypertension.

NREM total delta power was unrelated to BP or hypertensive status in cross-sectional analyses, regardless if it was age adjusted only or

fully adjusted. In the longitudinal age-adjusted analyses, lower NREM total delta power was associated with greater increases in DBP over time, adjusted for baseline DBP ($P = .01$), and tended to be associated with greater risk for incident hypertension (hazards ratio, .552 [95% CI, 0.39–1.03]). In the multivariate models, those with lower NREM delta power had a greater increase in DBP over the follow-up ($P = .006$) and tended to be at greater risk for incident hypertension ($P = .05$) (Table 2).

3.3. Other relevant analyses

In the cross-sectional analyses of BP, significant covariates were age, site, race/ethnicity, BP medications, BMI, and education. For hypertension prevalence, significant covariates were race/ethnicity, BMI, DM, and smoking. In the longitudinal analyses, significant covariates for BP change were baseline BP, BP medication use, age, BMI, education, and DM (for DBP only). For incident hypertension, significant covariates were BMI and DM.

Exploratory analyses examined if the type of BP medication (i.e., β blockers, diuretics, angiotensin-converting enzyme inhibitors, calcium channel blockers) influenced BP levels or sleep variables; no class of drugs was differentially impacted. Women who reported taking any BP medication at baseline had less efficient sleep ($P < .005$) and higher total beta power ($P = .006$).

4. Discussion

We hypothesized that sleep duration, efficiency, and NREM total delta and beta power would be related to an increased risk for hypertension and elevated BP in midlife women. At baseline hypertensive participants had shorter and less efficient sleep than normotensive participants in age-adjusted models. However, there were no differences in hypertensive status and no significant associations with BP in multivariate models. Blacks were at greater risk for hypertension and had shorter sleep duration and less efficiency sleep than whites and Chinese in the SWAN Sleep Study [17]. Thus it is not surprising that the addition of race/ethnicity only to the age-adjusted models resulted in nonsignificant results. We also examined if there were threshold effects of sleep duration, given that previous publications compared those with short sleep, (i.e., <5 or 6 h) to those with normal sleep. Again we did not observe an association.

On the other hand, higher total beta power was concurrently related to hypertensive status, which included antihypertensive medication in the definition but not to continuously measured BP, with antihypertensive medication as a covariate in the multivariate models. The difference in findings between these outcomes suggests that antihypertensive medications may impact NREM beta power. Indeed supplementary analyses showed that women who used antihypertensive medications had higher NREM beta power. On the other hand, lower NREM delta power was related to incident hypertension and to increases in DBP over time in multivariate models. The fact that the significant findings involved quantitative EEG sleep measures suggests that these measures may be important to include in future investigations of risk for hypertension.

Should the results for the EEG measures not be due to chance, it is important to consider plausible biologic explanations for the results. As summarized by Fung et al. [16], there was a fall in BP and heart rate during NREM sleep, particularly during SWS, considered to be due to decreased sympathetic activity and increased parasympathetic activity [25]. Conversely, lower SWS was associated with elevated nighttime BP, which is a risk factor for future hypertension and its complications [26]. Experimental studies of SWS deprivation show increases in nighttime BP relative to daytime BP in healthy volunteers [27]. Thus the impact of sleep depth may be through sleep-induced alterations of the relationship of the sympathetic and parasympathetic nervous system with nighttime BP.

Our study has a number of limitations. First, the sample size is relatively small for cohort studies of the determinants of hypertension, and the power was too low to detect ethnic differences in the pattern of results. Second, SWAN Sleep Study participants did not have measures of nighttime BP. Studies suggest that short inefficient sleep is particularly associated with elevated nighttime BP [28–30] and mechanisms connecting poor sleep and hypertension are likely to involve the absence of the beneficial fall of BP and heart rate at night.

Although the sample size was small, our study has a number of advantages. It used state of the art measures of sleep including NREM total beta and delta power measures. Sleep duration and efficiency were measured for two nights at the participant's home, which increases the validity of assessments and avoids the possibility of the first-night effect (i.e., poorer sleep on a first night of assessment due to the novelty of equipment). A substantial number of covariates were assessed. Finally, the sample comprised women of various ethnic groups who are known to have different patterns of sleep in our study and in other studies [17,31–33].

Sleep duration and efficiency measured by PSG were unrelated to high BP in midlife women in multivariate models. Quantitative EEG indices of sleep may be related to the risk for increasing BP in midlife and are worthy of future investigation in larger samples of men and women.

Funding sources

Funding for the SWAN Sleep Study is from the National Institute on Aging (Grants R01AG019360, R01AG019361, R01AG019362, R01AG019363). The Study of Women's Health Across the Nation (SWAN) has grant support from the National Institutes of Health, Department of Health and Human Services, through the National Institute on Aging, the National Institute of Nursing Research and the NIH Office of Research on Women's Health (Grants U01NR004061, U01AG012505, U01AG012535, U01AG012531, U01AG012539, U01AG012546, U01AG012553, U01AG012554, U01AG012495). Sleep data were processed with the support of RR024153 and analytic support was provided by R01HL04607. The content of this manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Aging, National Institute of Nursing Research, Office of Research on Women's Health or the National Institutes of Health.

Clinical Centers: University of Michigan, Ann Arbor, Siobán Harlow, PI 2011–present, MaryFran Sowers, PI 1994–2011; Massachusetts General Hospital, Boston, MA – Joel Finkelstein, PI 1999 – present; Robert Neer, PI 1994–1999; Rush University, Rush University Medical Center, Chicago, IL – Howard Kravitz, PI 2009 – present; Lynda Powell, PI 1994–2009; University of California, Davis/Kaiser – Ellen Gold, PI; University of California, Los Angeles – Gail Greendale, PI; Albert Einstein College of Medicine, Bronx, NY – Carol Derby, PI 2011 – present, Rachel Wildman, PI 2010–2011; Nanette Santoro, PI 2004–2010; University of Medicine and Dentistry – New Jersey Medical School, Newark – Gerson Weiss, PI 1994–2004; and the University of Pittsburgh, Pittsburgh, PA – Karen Matthews, PI.

NIH Program Office: National Institute on Aging, Bethesda, MD – Winifred Rossi 2012–present; Sherry Sherman 1994–2012; Marcia Ory 1994–2001; National Institute of Nursing Research, Bethesda, MD – Program Officers.

Central Laboratory: University of Michigan, Ann Arbor – Daniel McConnell (Central Ligand Assay Satellite Services).

Coordinating Center: University of Pittsburgh, Pittsburgh, PA – Maria Mori Brooks PI 2013–present; Kim Sutton-Tyrrell, PI 2001–2012; New England Research Institutes, Watertown, MA – Sonja McKinlay, PI 1995–2001.

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Conflict of interest

Dr. Buysse has served as a paid consultant on scientific advisory boards for the following companies: Esai, Merck, Philips Respironics, Purdue Pharma and General Sleep Corporation. Dr. Buysse has also spoken at single-sponsored educational meetings for Servier. He has also spoken at a single-sponsored lecture for Astellas. Dr. Hall has given lectures on sleep in women to the National Sleep Foundation. No other authors have conflicts to disclose.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2013.11.002.

Acknowledgment

We thank the study staff at each site and all the women who participated in SWAN Sleep Ancillary Study.

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